CARDIOMEGALY AND GENERALIZED OEDEMA DUE TO VITAMIN C DEFICIENCY

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SYNOPSIS

A case of Vitamin C deficiency with massive oedema of the legs, ascites, pleural effusion and cardiac enlargement is described. The response to Vitamin C therapy was prompt and remarkable. Some rare and interesting manifestations of scurvy are described and the possible mechanism for the cardiomegaly and oedema is discussed.

INTRODUCTION

The clinical manifestations of scurvy both in infants and adults are well known. The less common manifestations like generalized oedema and cardiac enlargement are not so well documented. Hodges, R.E. (1971) listed out 10 uncommon ways that Vitamin C deficiency can present clinically.

The French explorer Jacques Cartier first described sudden death due to myocardial involvement in scurvy. Darling and others (Tropical Diseases—Manson-Bahr) had observed that the mine workers on the Rand in Africa developed an atypical type of scurvy where the heart underwent primary hypertrophy and subsequently dilatation. According to Spodick, D.H. (1970) haemopericardium due to Vitamin C deficiency was apparently known in ancient times and was described in great detail over a century ago. Hood, J. (1970) in reference to a Vitamin C deprivation experiment carried out in 1944-46 by Bartley, W. *et al* points out that 2 out of the 10 subjects developed E.C.G. changes such as prolonged PR & ST elevation. Sament, S. (1970) observed E.C.G. changes such as prolonged PR, ST elevation in leads I & II, and sudden death, due probably to myocardial involvement, in African populations and return of the E.C.G. to normal after therapy.

We describe a case of scurvy with generalized oedema and cardiac enlargement.

CASE REPORT

A Chinese female, OAW, aged 40 presented with a history of insidious onset of swelling of the legs associated with exertional dyspnoea for 6 days prior to admission. In the past 6 years she had been attending psychiatric clinic and was on phenothiazine.

On admission she was found to be pale with a slightly puffy face. She had gross oedema of the lower limbs with multiple petechiae, some of which were peri-follicular. The hair on the lower limbs was scanty but appeared coiled. The pulse rate was 70 per minute with the B.P. 120/80. Heart was normal. Chest revealed bilateral pleural effusion. The liver was 3 cm. below the sub-costal margin with free fluid present in the abdomen. There were no spider naevi or liver palms. The neck veins were not engorged. The gums were spongy, septic and bleeding. Mentally she was slow, dull and apathetic.

Investigations done on admission:—

Hb. 9·9 gm. %: T.W. 6,900/cmm.: Platelets: 355,000/cmm.

E.S.R. 16 mm./hour. Hess's test was negative.

LE cells test: negative: Anti-nuclear factor: negative.

Serum Electrolytes:	K. 4·9 mEq./litre.
	Na. 136 mEq./litre.
	Cl. 107 mEq./litre.
Serum proteins:	Albumin: 3·8 gm.%
	Globulins: 1·5 gm.%
Serum cholesterol:	110 mgm./100 ml.
Urine FE & ME nad:	Blood urea 20 mgm.%
Serum Pyruvic acid:	1 mgm.%
(Normal < 1 mgm.%)	

X-ray chest: enlarged heart with bilateral pleural effusion.

Minimal opacity left upper zone: (Fig. 1).

Mantoux test: 15 mm. (positive).

E.C.G. within normal limits. (Fig. 4).

Right pleural tap yielded slightly straw coloured fluid with a protein content of 1·8 gm.%. There were a few red blood cells and lymphocytes. No organisms seen. Cultures of pleural fluid and laryngeal swabs were negative for acid fast bacilli.

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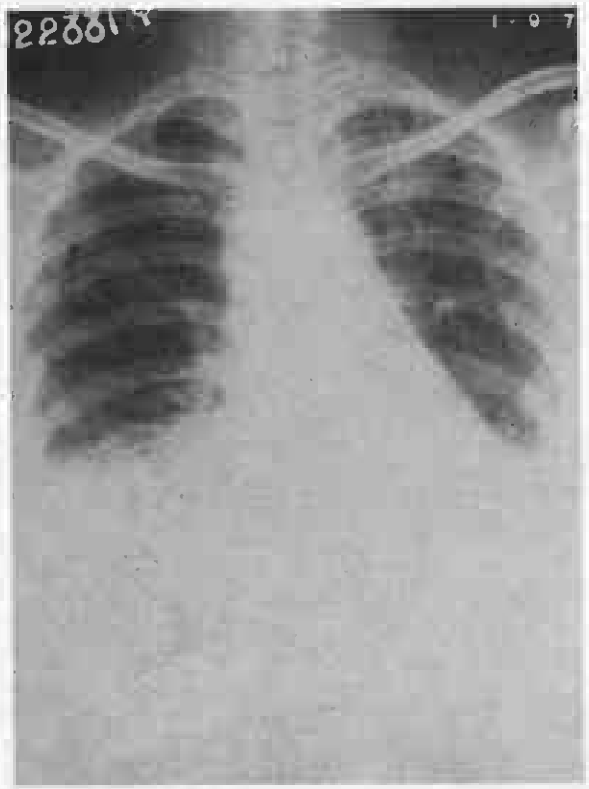

Fig. 1. On 1.9.72— Date of admission. Bilateral pleural effusion with cardiomegaly. Minimal opacity left upper zone.

Vitamin C loading test done on 2 consecutive mornings by giving 500 mgm. of Vitamin C (70 mgm./stone body weight) at 7 a.m. each morning and then estimating the amount excreted in the specimen of urine passed between 11 a.m. to 1 p.m. (Maximum excretion occurs between 4-6 hours after intake) each day, gave the following results.

1st day specimen < 10 mgm.%
2nd day specimen < 1 mgm.%
(Normal: > 50 mgm.%)

A diagnosis of Vitamin C deficiency with generalised oedema, cardiomegaly and possible minimal Koch's left upper zone was made. Patient was started on oral Vitamin C 400 mgm. daily when her clinical condition was about the same as on admission. X-ray chest still showed a large heart with pleural effusion (Fig. 2). Meanwhile she was still taking her phenothiazine. Nothing else was given from the day of admission other than a normal ward diet.

Within 48 hours her petechiae had begun to disappear and the oedema was substantially less. She made remarkable progress and within a week the ascites, oedema and petechiae had completely disappeared. X-ray chest done 14 days after starting treatment (Fig. 3) showed complete clearance of the pleural effusion with a normal size heart. There was minimal fibrotic lesion LUZ. Her mental state showed some improvement. She was more cheerful, active and co-operative.

DISCUSSION

The diagnosis of Vitamin C deficiency is made on clinical grounds and by the estimation of plasma ascorbic acid or by the ascorbic acid saturation test. Of these plasma ascorbic acid estimation is more reliable (Bartley and others, 1953).

Our patient had several clinical features of scurvy such as peri-follicular bleeding, which is almost pathognomonic (Hodges, R. E., 1971), hypertrophic spongy gums and coiled hair. These are non-specific and late manifestations.

Hodges, R. E. (1971) in a Vitamin C depletion experiment on 5 subjects, described some uncommon and very interesting clinical manifestations such as ocular haemorrhages, Sjögren's syndrome, femoral neuropathy from bleeding into the femoral sheath, oliguria with oedema of the lower limbs, psychological disturbances like hysteria and depression, impaired vascular reactivity and scorbutic arthritis which is clinically similar to Rheumatoid arthritis.

Our patient had two uncommon and interesting manifestations namely cardiac enlargement and massive oedema of the lower limbs with ascites and bilateral pleural effusion. Hodges, R. E. (1971) makes no mention of ascites, pleural effusion, cardiomegaly, E.C.G. or X-ray chest changes in any of his subjects even though 2 of them complained of dyspnoea on exertion just as our patient did.

Changes in the E.C.G. due to scurvy have been recorded (Hood, J., 1970; Sament, S., 1970). In spite of the cardiac enlargement, the E.C.G. of our patient was normal.

The clinical manifestations of experimentally induced scurvy and spontaneous scurvy are very similar (Hodges, R. E., 1971). Whether the manifestations we have described depend upon the length and degree of deprivation, or on other factors such as climate, activity, racial predisposition or any other associated illness, we are unable to say. However, spontaneous scurvy generally is more severe and involves more systems.

Other causes for the cardiac enlargement were considered and ruled out. There was no evidence of hypertensive heart disease, ischaemic heart disease or valvular disease. There was no evidence of cardiac beri-beri; the serum pyruvic acid was normal and there were no other clinical signs such as hyperkinetic circulation, wide pulse pressure, gallop rhythm with tachycardia or peripheral neuropathy to suggest B1 deficiency.

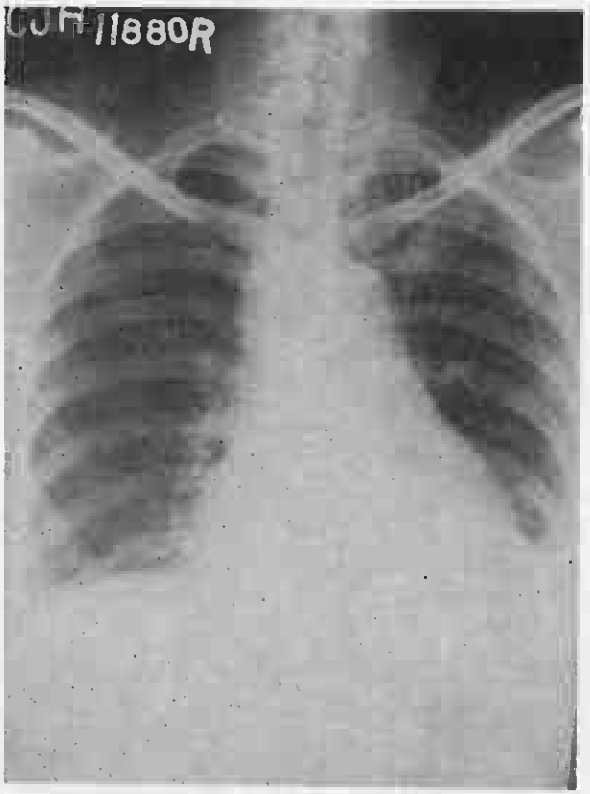

Fig. 2. On 5.9.72 after right pleural tap, and one day before starting Vitamin C. Effusion on right less following tap. Otherwise no significant change.

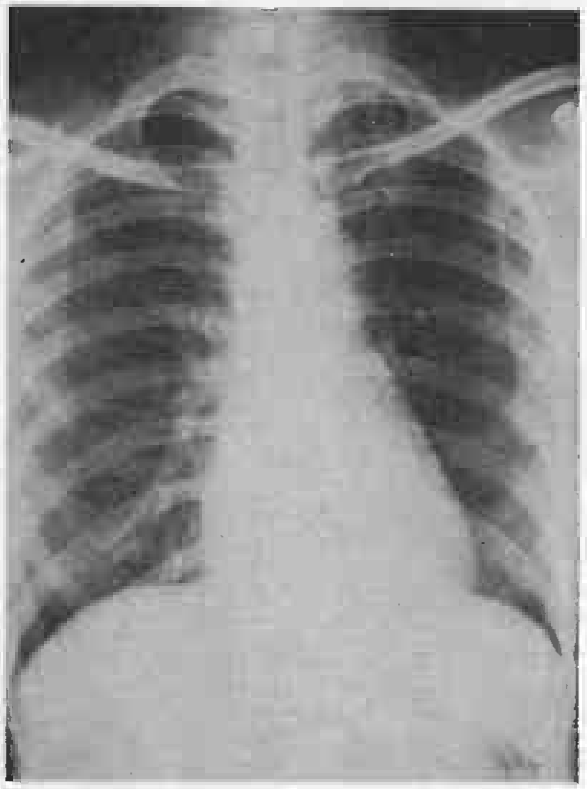

Fig. 3. On 20.2.72. After 2 weeks of Vitamin C. No pleural effusion. Heart size normal. Fibrotic lesion left upper zone.

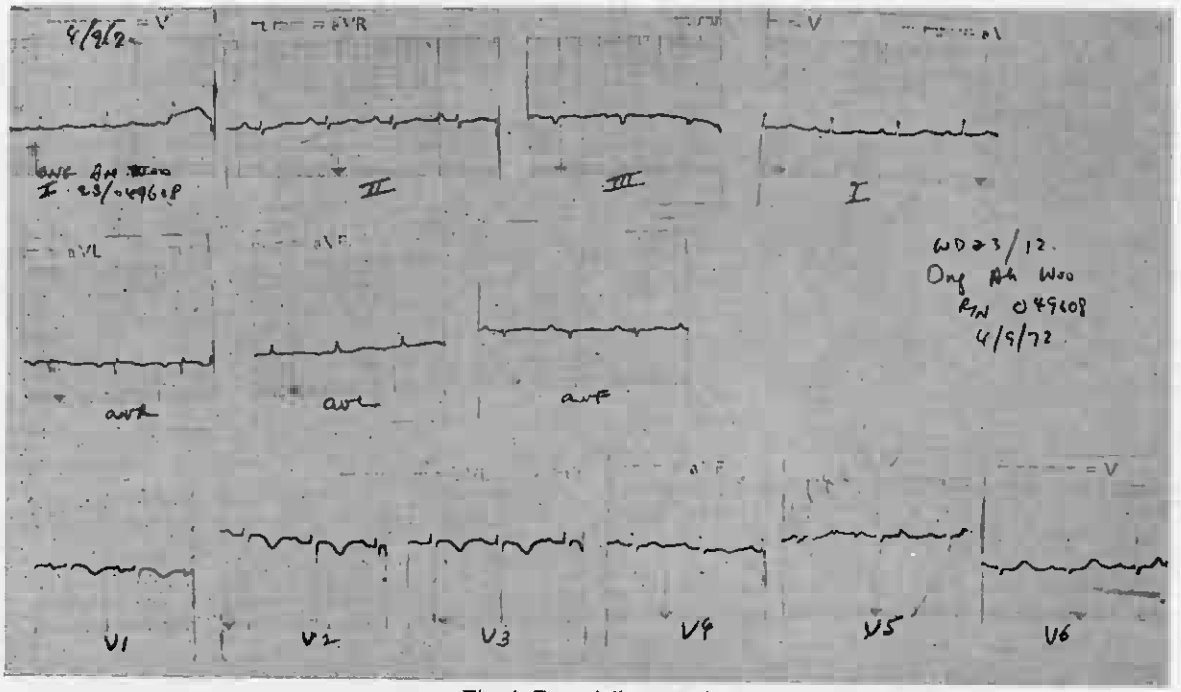

Fig. 4. Essentially normal.

Phenothiazine which the patient was on has been reported to cause myocardial lesion with E.C.G. changes (Richardson, H.L. *et al*, 1967) but our patient continued to take her phenothiazine during her stay in the hospital and the heart size returned to normal after Vitamin C therapy.

There was no clinical, radiological or E.C.G. evidence to suggest pericardial effusion but that the cardiac enlargement was due to an effusion cannot be ruled out even though the return of the heart size to normal within 14 days after therapy would favour cardiac dilation and/or hypertrophy.

All other causes of generalized oedema were looked into and excluded. The serum proteins were normal. The patient was not in cardiac failure. There was no evidence of hepatic or renal disease. Moreover, the response to Vitamin C was dramatic and conclusive. The oedema from the lower limbs began to clear within 48 hours. The ascites had cleared by the 7th day and the chest X-ray done on the 14th day showed complete clearance of pleural effusion with a normal size heart and a minimal fibrotic lesion in the left upper zone.

Her mental dullness and apathy showed some improvement after treatment. She became more cheerful, alert and active, as has been observed by others (Scobie, B.A., 1969).

The mechanism of action of ascorbic acid remains obscure. Vitamin C depletion causes oliguria with fluid retention. On giving ascorbic acid a prompt diuresis occurs with clearing of the oedema. The diuretic effect has been observed by Hodges, R.E. (1971) and others. Since Vitamin C is essential for maintaining the integrity of the vascular system, increased capillary permeability probably contributes towards the leakage of fluid from the vascular compartment.

Whether the cardiomegaly is due to pericardial effusion, primary myocardial involvement or to hypervolemia following oliguria and fluid retention or is due to all 3 factors and some other unknown mechanism is still speculative.

CONCLUSION

We describe a case of scurvy with cardiomegaly and generalized oedema. Response to Vitamin C was dramatic and remarkable.

Some uncommon manifestations of scurvy are described and the mechanism for the cardiomegaly and generalized oedema is briefly discussed.

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